In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 16-998V
Filed: March 1, 2023
PUBLISHED

ARTHA TIMOTHY, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Horner Trigeminal Neuralgia; Tic Convulsif; Hemifacial Spasms; Influenza ("Flu") Vaccine; Ruling on the Record

Leigh Finfer, Muller Brazil LLP, Dresher, PA, for petitioner.
Ryan Pyles, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On August 12, 2016, petitioner, Artha Timothy, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),[2] alleging that her receipt of an influenza ("flu") vaccination on October 9, 2014, caused the development of her trigeminal neuralgia. (ECF No. 1.) For the reasons set forth below, I conclude that petitioner is not entitled to an award of compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute;

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

[2] All references to "§ 300aa" below refer to the relevant section of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a causal link between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, petitioners may show that they suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. In such cases, the Table Injury is presumed to have been caused by the vaccine. § 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

In many cases, however, the vaccine recipient may have suffered an injury not covered by the Vaccine Injury Table. In these "off-table" cases, an alternative means exists to demonstrate entitlement to a Program award. The petitioner may demonstrate entitlement by showing that the recipient's injury was "caused-in-fact" by the vaccine they received, a showing often referred to as "actual causation." § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In off-table cases, the presumptions available under the Vaccine Injury Table are inoperative, and the burden is on the petitioner to introduce evidence demonstrating that the vaccination was responsible for the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

To show actual causation, petitioner must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination caused the alleged injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition, but must demonstrate that the vaccination was a "substantial factor" and a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). This standard has been interpreted to require "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;" the logical sequence must be supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal

> relationship between vaccination and injury. If Althen satisfies this burden, she is "entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine."

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court explained that petitioners are not required to provide medical literature supporting their theory of causation so long as they supply the medical opinion of an expert. *Id*. at 1279-80. The *Althen* court also indicated that Program fact finders may rely upon "circumstantial evidence" to determine causation, a standard it held to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." *Id*. at 1280.

In this case, petitioner has alleged that the influenza vaccine caused her to suffer trigeminal neuralgia. Because trigeminal neuralgia is not listed on the Vaccine Injury Table, petitioner must satisfy the above-described *Althen* test for establishing causation-in-fact.

II. Procedural History

Petitioner filed her petition, medical records, and statement of completion, on August 12, 2016. (ECF Nos. 1, 3; Exs 1-6.) The case was initially assigned to Special Master Hamilton-Fieldman. (ECF No. 4.) Petitioner subsequently filed additional medical records on October 19 (Exs. 5-7) and November 17, 2016 (Ex. 8). (ECF Nos. 8, 10.) On January 9, 2017, the case was reassigned to Special Master Sanders. (ECF No. 14.)

Thereafter respondent filed a Rule 4(c) report arguing that the evidence presented did not meet petitioner's burden and recommending against compensation. (ECF No. 16.) After a Rule 5 status conference was held on March 15, 2017, Special Master Sanders ordered petitioner to file an expert report on causation. (ECF No. 17.) On April 7, 2017, petitioner filed transcribed pages from her EliteCare medical records. (ECF No. 18, Ex. 9.) On June 15, 2017, petitioner filed her first expert report from Dr. Eric Gershwin (immunology). (ECF No. 21, Ex. 10.) On September 13, 2017, respondent filed expert reports from Dr. Subramaniam Sriram (neurology, microbiology immunology) (Ex. A) and Dr. Neil Romberg (allergy / clinical immunology) (Ex. C). (ECF Nos. 23-25.) The parties subsequently filed additional expert reports from Drs. Gershwin, Sriram, and Romberg on October 20th and December 19, 2017. (ECF Nos. 26-27; Ex. 11; Exs. DD, EE.)

During a status conference held on January 17, 2018, Special Master Sanders expressed concern that petitioner's expert was relying primarily on literature relating to Bell's Palsy whereas petitioner's claim was for trigeminal neuralgia. She ordered petitioner to file a supplemental expert report explaining "how Bell's Palsy and trigeminal neuralgia are related, including why the two conditions are medically comparable both generally and in support of a theory of causation relating the flu vaccination to trigeminal neuralgia" and how "a vaccine injected into Petitioner's deltoid can cause a localized

reaction in her trigeminal nerve." (ECF No. 28.) Subsequently the parties filed supplemental expert reports from Dr. Gershwin and Drs. Sriram and Romberg, respectively. (ECF Nos. 29, 31; Ex. 12; Exs. GG, HH.) Concurrently, petitioner filed medical literature, including a translated copy of one of Dr. Gershwin's references. (ECF No. 29, Ex. 12.2.)

After a status conference on June 18, 2018, Special Master Sanders noted that Dr. Gershwin's second supplemental expert report "did not adequately answer the questions outlined in the [] January 17, 2018 Order." (ECF No. 33.) Special Master Sanders ordered petitioner to file "an article that can further explain Petitioner's causation theory" and if necessary, commentary from Dr. Gershwin. (ECF No. 33.) On July 23, 2018, petitioner filed the requested medical literature. (ECF No. 34, Exs. 13-17.) On October 9, 2018, respondent filed Dr. Romberg's final expert report. (ECF No. 35, Ex. JJ.) Petitioner filed updated medical records on December 13, 2018 and May 29, 2019. (ECF Nos. 37-39, Ex. 18-20.)

The case was reassigned to my docket on March 2, 2021. (ECF No. 42.) On April 5, 2021, the parties filed a joint status report indicating that the case was ripe for a hearing. (ECF No. 43.) To accommodate the availability of the witnesses, a one-day fact hearing was set for October 1, 2021, and a two-day entitlement hearing was set for November 4, 2021. (ECF No. 47.) However, on September 29, 2021, a status conference was held with the parties and petitioner's counsel indicated that she spoke with petitioner's adult daughter who informed her that petitioner had become incapacitated due to a stroke. (ECF No. 58.)[3] Accordingly, the fact hearing and entitlement hearing were cancelled. (*Id.*) On March 18, 2022, the parties filed a joint status report proposing to instead proceed with briefing on the record. (ECF No. 65.)

On April 13, 2022, petitioner filed further updated medical records and a statement of completion. (ECF Nos. 66, 67.) On May 20, 2022, petitioner filed her motion for a ruling on the record. (ECF No. 68.) That same day, respondent filed his cross-motion and memorandum. (ECF No. 69.) No reply briefs were filed.

I have determined that the parties have had a full and fair opportunity to present their cases and that it is appropriate to resolve this issue without a hearing. See Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Sec'y of Health & Human Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record."). Accordingly, this matter is now ripe for resolution.

[3] No affidavit by petitioner has been filed in this case as required by Vaccine Rule 2(c)(2). However, given the outcome of this decision, it is not necessary to remedy that deficiency.

III. Factual History

a. As Reflected by the Medical Records

i. Pre-Vaccination Records

Petitioner was born July 16, 1953, and her prior medical history is significant for hypertension, gastroesophageal reflux disease ("GERD"), premature atrial contractions, high cholesterol, type 2 diabetes mellitus, and sinusitis. (Ex. 3, p. 20; Ex. 5, p. 8.) Petitioner's past surgical history includes a hysterectomy, neck surgery, neuroplasty decompression of the median nerve (carpal tunnel), and trigger point injections. (Ex. 3, p. 20.) Petitioner reported that she was diagnosed with migraine headaches in the 1980's and underwent temporomandibular joint ("TMJ") surgery to provide relief from the headaches. (Ex. 6, p. 20.) It was later discovered that she had a "disk problem in her neck," and once that issue was addressed "she no longer had headaches." (*Id.*) It appears that petitioner underwent TMJ surgery in 1990, but it is unclear when her headaches ceased. (Ex. 6, p. 95.)

On May 6, 2013, petitioner presented to Lakeview Urgent Care complaining of her "wors[t] headache ever." (Ex. 5, pp. 45, 54.) That same day a CT scan of petitioner's brain was taken without contrast. (*Id.* at 50.) The results were unremarkable and multiple CT levels were obtained from the posterior fossa to the vertex that showed "no areas of abnormal density." (*Id.*) On May 7, 2013, petitioner refused a lumbar puncture procedure "because of the pain." (*Id.* at 44.) Petitioner was discharged May 8, 2013 and referred to a neurologist for further evaluation. (*Id*. at 29-30.)

On June 28, 2013, petitioner presented to Lakeview Urgent complaining of headaches, "this time different with right sided visual change, 'like looking through a screen.'" (Ex. 5, p. 8.) That same day petitioner underwent a CT scan of her head without contrast which revealed sinus inflammation and no acute intracranial abnormalities. (*Id.* at 13.) Petitioner was discharged June 30, 2013. (*Id.* at 7.)

On October 9, 2014, petitioner reported to Elite Care of Fayetteville for a medication refill and a flu vaccine. (Ex. 2, p. 24, Ex. 9, p. 5.) At this visit petitioner reported a history of present illness which included high blood sugar, urinary incontinence, and lower back pain for four days without specific trauma. (Ex. 2, p. 24, Ex. 9, p. 5.) Her diagnoses include diabetes mellitus, hypertension, myofascial strain, urinary incontinence, and GERD. (Ex. 9, p. 5.) Petitioner's vaccine record confirms that she received an influenza vaccination on October 9, 2014[4]. (Ex. 1.)

[4] The printed vaccination form lists "2014" Pneumonia Shots," though "Pneumonia" is crossed out and "Flu" is handwritten below. (Ex. 1.)

i. **Post-Vaccination Records**

On October 24, 2014, petitioner reported to PA-C Travis Ross complaining of pain in the left side of her face beginning on October 12, 2014, as well as headaches on her left side. (Ex. 2, p. 16.) She complained of "sharp/shooting" pain that lasted for several seconds. (Ex. 9, p. 4.)[5] Petitioner reported that touching certain areas and "gripping teeth" aggravated her pain – which distributed to her nose and teeth. (*Id.*) The notes from this visit indicate that PA-C Ross "suspect[ed] tic douloureux."[6] (*Id.*) Petitioner was diagnosed with "facial pain." (*Id.*)

On December 8, 2014, petitioner returned to her physician assistant for a refill of her prescriptions and to "follow up w[ith] face." (Ex. 2, p. 12; Ex. 9, p. 3.) While petitioner noted that "facial pain has improved," she noted "still 'shock' if [she] touch[es] [her] face in [the] maxillary region." (Ex. 9, p. 3.) The notes section indicates "[d]iscussed need for compliance with ace inhibitor, [r]eviewed DNA (renaissance testing), [and] ? tic douloureux." (*Id.*) Petitioner was diagnosed with facial pain and hypertension and referred for a neurology consultation. (*Id.*)

On December 15, 2014, petitioner reported to her cardiologist Brenda DePaola, D.O., for a follow-up visit regarding fatigue, shortness of breath, and her heart health. (Ex. 3, pp. 9-12.) Petitioner reported that she had suffered five "episodes" since her last visit, though "only a couple that were real intense" and most were "brief and short lived." (Ex. 3, p. 9.) Among her "active problems" were dizziness, GERD, hypertension, and trigeminal neuralgia. (*Id.*) Dr. DePaola ordered an exercise nuclear stress study for further evaluation.[7] (*Id.* at 12.)

On December 29, 2014, petitioner presented to neurologist Venugopal Gadipudi, M.D., for an initial evaluation. (Ex. 4, p. 5.) In the history of present illness, Dr. Gadipudi noted petitioner was experiencing "left facial spasms" and "pain around the eye." (*Id.*) Petitioner rated her pain at "20/10" and indicated that her pain increased while washing her face since October 2014. (*Id.*) Petitioner did not recall any infection and denied having any visual or speech disturbances. (*Id.*) In his assessment Dr.

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[5] Exhibit 9 contains transcribed pages from Elite Care, Travis Ross, PA-C. (*See* ECF No. 66-1.)

[6] "Tic douloureux" is another term for trigeminal neuralgia. *Tic douloureux,* DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=113729&searchterm=tic+douloureux (last accessed Feb. 27, 2023). When accompanied by hemi facial spasms, the syndrome is known as "tic convulsif." (E.T. Fonoff et al., *Neurovascular compression in painful tic convulsif*, 151 ACTA NEUROCHIR. 989 (2009) (Ex. H).)

[7] In her petition, petitioner states that she presented to Dr. DePaola "with ongoing pain and numbness in the left side of her face" and "Dr. DePaola diagnosed Petitioner with trigeminal neuralgia," however this appears to overstate the medical record. (ECF No. 1, ¶5; s*ee id.* at 9-12.) Trigeminal neuralgia is listed as an "active problem." (*Id.* at 9.) However, nothing in the medical records confirms that it, or its symptoms, were discussed during the encounter. There is no discussion of relevant symptoms in either the history of present illness or review of systems. No relevant physical exam was documented and trigeminal neuralgia was not included in either Dr. DePaola's assessment or plan. (Ex. 3, pp. 9-12.)

Gadipudi noted that petitioner "had a flu shot 3 to 4 days before the acute onset of pain" and that the pain was "episodic in nature[,] suggestive of trigeminal neuralgia." (*Id.* at 6.) Dr. Gadipudi recommended an MRI of the brain and a follow-up visit in one month. (*Id.*)

On January 12, 2015, petitioner underwent an MRI. (Ex. 4, p. 7.) The reason for the MRI exam listed "multiple sclerosis" and the "additional history" section of the diagnostic test indicates that petitioner had a history of multiple sclerosis, although no other records indicate that petitioner was diagnosed with multiple sclerosis. (*Id.*) The MRI revealed a mild degree of generalized cortical atrophy, multifocal areas of increased signal intensity, and subcortical white-matter regions bilaterally -- which can be seen with "demyelinating plaques of multiple sclerosis" and "chronic small-vessel ischemic changes." (*Id.*) However, the results indicated that "[n]one of these demonstrate abnormal contrast enhancement…to suggest active demyelination at this time." (*Id.*) Petitioner's MRI also revealed mild inflammatory sinus disease, which was labeled as "probably chronic." (*Id.*)

On February 6, 2015, petitioner returned for a follow-up visit with Dr. Gadipudi. (Ex. 4, p. 4.) Dr. Gadipudi noted she "did not have as many spasms today" but below in his assessment remarked that "she currently does not have spasms." (*Id.*) He also "explained that the flu shot would not have any relation to the facial spasms." (*Id.*) Dr. Gadipudi also noted petitioner's past significant sinus disease. (*Id.*) He ordered petitioner to follow-up in two months, and to continue antibiotics.[8]

On April 9, 2015, petitioner presented to her physician assistant for a medication refill. (Ex. 2, p. 10.) The notes indicate that petitioner and her physician assistant "discussed trigeminal neuralgia," though her diagnoses listed only diabetes, hypertension, and GERD. (Ex. 9, p. 2.) Petitioner refused a statin and was advised to "return for lipid as needed." (*Id.*)

On July 7, 2015, petitioner presented to Dr. DePaola for a follow-up cardiology visit. (Ex. 3, pp. 5-8.) Dr. DePaola notes that petitioner did not have "too many issues with recurrent arrythmia[,] [s]he has about 3 episodes per month." (*Id.* at 5.) Dr. DePaola remarked that "[f]rom a cardiac standpoint, she is doing well." (*Id.* at 8.) Petitioner in her petition states that she "followed up with Dr. Brenda DePaola with ongoing pain and numbness in the left side of her face." (ECF No. 1, ¶ 9.) These symptoms are not reflected in the medical records from Dr. DePaola. (*See* Ex. 3, pp. 5-8.) It appears that petitioner saw Dr. DePaola regarding her heart conditions, though again trigeminal neuralgia was listed as one of petitioner's "active problems." (*Id.*)

On July 15, 2015, petitioner presented to Dr. Gadipudi with chief complaints of left facial spasms and trigeminal neuralgia. (Ex. 4, p. 3.) He noted her history of

[8] As of this visit, petitioner's current medications listed Coreg, Aspirin, and Metformin. (Ex. 4, p.4.) It is unclear from the records what antibiotics petitioner was taking, when she began taking them, or why she was taking them. In her status report from January 9, 2017, petitioner states that "antibiotics were prescribed during an October 24, 2014 primary care visit." (ECF No. 13.)

trigeminal neuralgia, diabetic neuropathy, and migraine headaches (unrelated to trigeminal neuralgia). (*Id.*) Dr. Gadipudi also noted that petitioner "ha[d] some intermittent breakthrough paresthesias, which were improved on Tegretol." (*Id.*) Dr. Gadipudi's notes do not indicate whether petitioner's paresthesia was caused by her trigeminal neuralgia or her diabetic neuropathy. (*See id.*) He ordered petitioner to continue taking Tegretol and to follow-up in three months. (*Id.*)

On November 12, 2015, petitioner presented to Travis Ross, PA-C, with chief complaints of hearing loss and diabetes follow-up care. (Ex. 2, p. 6.) Petitioner described pressure and pain in her left ear with a loss of hearing that was "moderately severe." (*Id.*) She also noted "the problem started gradually, 6 months [prior]," although there was also "a previous occurrence of similar symptoms." (*Id.*) At the time petitioner had no diabetes-related symptoms. (*Id.*) There was no mention of trigeminal neuralgia or paresthesia during this visit; and her past medical history listed only arthritis, diabetes, and GERD. (*Id.*) Petitioner's Metformin was decreased to 500 mg twice daily and she was also referred to an ENT. (*Id.* at 8.)

On November 17, 2015, petitioner returned to Dr. Gadipudi for a follow-up visit. (Ex. 4, p. 1.) Petitioner was instructed to continue taking Tegretol as well as Neurontin, given her history of trigeminal neuralgia, diabetic neuropathy and migraine headaches. (*Id.*) Petitioner was scheduled to return for a follow-up in one to two months. (*Id.*)

On January 7, 2016, petitioner reported to Dr. DePaola for another cardiology follow-up, noting prior sinus bradycardia, atrial fibrillation, and acid reflux. (Ex. 3, pp. 1-4.) Dr. DePaola encouraged petitioner to report any further episodes of dizziness so that she could schedule a monitor to "ensure she isn't profoundly bradycardic."[9] (Ex. 3, p. 4.)

On January 29, 2016, petitioner reported to the emergency department complaining of headaches and transient confusion. (Ex. 6, pp. 14, 17.) Petitioner described blurred vision with visualization of dark spots. (*Id.* at 16.) There was concern for transient ischemic attack and petitioner was transferred to Moore Regional Hospital for further attention. (*Id.* at 14.) Petitioner was seen by neurologist Nicole Odom, M.D. (*Id.* at 20-23.) Dr. Odom noted symptoms of left hand and arm numbness up to the shoulder, confusion, difficulty breathing, and a headache – all of which gradually subsided. (*Id.* at 19.) Petitioner also described experiencing similar symptoms 2-3 years prior, including the "worst headache of her life." (*Id.*) There was no discussion of facial spasms or numbness during this visit, though "tic douloureux" was listed among petitioner's discharge diagnoses. (*Id.* at 14.)

Petitioner underwent a CT scan of her head on January 29, 2016, which revealed mild chronic paranasal sinus disease and otherwise "no acute intracranial process." (Ex. 6, p. 73.) On January 30, 2016, a brain MRI showed no acute ischemic

[9] The petition states that petitioner followed up with Dr. DePaola for "pain and numbness on the left side of her face" and that she had "informed Dr. DePaola that her pain had slightly decreased," though these symptoms do not appear in the notes from this visit. (ECF No. 1, ¶ 13; *but see* Ex. 3, pp. 1-4.)

event; advanced small vessel change; and scattered chronic paranasal sinus disease and bilateral chronic mastoid sinus disease. (*Id.* at 74.) Dr. Odom reviewed petitioner's MRI, finding it was notable for periventricular white matter disease but showed no evidence of acute stroke. (*Id.* at 22.) Dr. Odom found that petitioner's history was most consistent with transient ischemic attack. (*Id.*) A carotid ultrasound found no significant plaque bilaterally. (*Id.* at 14, 75.) An echocardiogram was performed which showed an ejection fraction of greater than 65%, no patent foramen ovale, and no obvious embolic source. (*Id.* at 14, 78-79.) Petitioner was discharged on January 30, 2016, with instructions to start taking Eliquis and to follow up with PA-C Ross and Dr. Odom. (*Id.* at 14-15.)

On March 9, 2016, petitioner presented to her physician assistant for a refill of her prescriptions. (Ex. 2, p. 2.) Her physical exam was normal and she was assessed with controlled type 2 diabetes. (*Id.* at 4.) PA-Ross planned to stop petitioner's metformin while monitoring her glucose and A1C. (*Id.*)

Petitioner returned to her neurologist Dr. Gadipudi on April 1, 2016, and again on June 6, 2016, who recommended that she continue taking Tegretol as well as Neurontin. (Ex. 4, p. 2, Ex. 8, p. 1.) On October 21, 2016, petitioner saw Dr. Gadipudi with new complaints of right-hand weakness and dropping a cup of coffee, as well as visual disturbances. (Ex. 18, p. 6.) Dr. Gadipudi also noted petitioner's "intermittent breakthrough pain." (*Id.*) As a result, Dr. Gadipudi started petitioner on Plavix, discontinued Aspirin, added Cymbalta in addition to the Neurontin, discontinued Tegretol, and scheduled a one-month follow-up. (*Id.*)

On November 3, 2016, petitioner underwent a CT scan of her brain after her hospitalization and transient ischemic attack. (Ex. 8, p. 8.) The CT scan revealed mild decreased attenuation of the white matter of each cerebral hemisphere "likely related to microvascular ischemic change and stable from prior MRI of the brain." (*Id.*) On December 2, 2016, petitioner returned to Dr. Gadipudi who concluded her CT scan was "unremarkable for any strokes." (Ex. 18, p. 5) He continued petitioner on Plavix, Neurontin, and ordered a follow-up in one to two months. (*Id.*) Petitioner continued her follow-up visits with Dr. Gadipudi for her trigeminal neuralgia throughout 2017 and 2018. (Ex. 18, pp. 2-4.)

On November 7, 2018, petitioner presented to Dr. Gadipudi for a follow-up visit. (Ex. 20, p. 4.) The progress note doesn't list any new symptoms, though Dr. Gadipudi added a prescription for 5 mg of Flexeril "for muscle spasms." (*Id.*) Then on November 9, 2018, petitioner underwent further MRI of her brain to evaluate her for demyelinating disease or other abnormalities. (*Id.* at 5.) Results of that scan showed no evidence of active demyelination and no evidence of intracranial hemorrhage, mass, edema, or restricted diffusion. (*Id.* at 5-6.)

Petitioner continued follow-up visits with Dr. Gadipudi through November of 2019. (Ex. 20, pp. 2-3; Ex. 21, p. 2.) Petitioner continued complaints of trigeminal neuralgia and headaches. (*Id.*) On May 6, 2019, Dr. Gadipudi noted petitioner was on

"Tegretol prophylaxis and Flexeril pm for headaches." (*Id.; see also* Ex. 21, p. 2 (11/1/2019 visit) (HPI, assessment, and plan same as 5/6/19 visit).) On May 17, 2019, petitioner presented to Travis Ross, PA-C for a diabetes follow-up. (Ex. 19, p. 5.) Petitioner did not have any diabetes-related symptoms, and the progress note does not mention facial spasms or trigeminal neuralgia. (*Id.*)

No records for Dr. Gadipudi were filed for 2020 or 2021. Petitioner's recent history is significant for a cerebrovascular accident in June 2021. On June 30, 2021, petitioner presented to PA-C Ross in follow-up after hospitalization.[10] (Ex. 22, pp. 8-10.) Petitioner's daughter reported a decrease in petitioner's cognitive skills. (*Id.* at 9.) Petitioner's physical exam was otherwise normal. (*Id.* at 9-10.) She was assessed with a recent cerebrovascular accident. (*Id.* at 10.) On September 9, 2021, petitioner returned to PA-C Ross whose assessment was controlled type 2 diabetes and cognitive deficit status post cerebrovascular accident. (*Id.* at 6.)

IV. Expert Opinions

a. Petitioner's Expert, M. Eric Gershwin, M.D.

Dr. Gershwin received his medical degree from Stanford University School of Medicine in 1971.[11] Dr. Gershwin is currently a Distinguished Professor of Medicine and Chief of the Division of Rheumatology, Allergy and Clinical Immunology at the University of California School of Medicine at Davis. (Ex. 10.) He has treated patients with neuroimmunologic disorders for over 40 years. (*Id.*) His career has been devoted to immunology, specifically the mechanisms that lead to breach of tolerance, including published works on genetic susceptibility, extensive studies of environmental factors, and work involving innate and adaptive immune responses. (*Id.*) He is board certified in internal medicine, allergy and immunology, and rheumatology. (*Id.*) Dr. Gershwin has published nearly 1,000 books, experimental papers, book reviews, and book chapters in the field of autoimmunity since 1967.

Dr. Gershwin explains that trigeminal neuralgia is principally a neuropathy of one or more branches of the trigeminal nerve. (Ex. 10, p. 2.) The disorder is common in older individuals and occurs often on one side of the body. (*Id.*) According to Dr. Gershwin, the etiology of trigeminal neuralgia is "enigmatic," and "there is no obvious etiology." (*Id.*) Dr. Gershwin likens trigeminal neuralgia to the mechanisms associated

[10] No hospitalization records have been filed, but respondent indicates he "does not view them as necessary for the resolution of this claim, as the focus would have clearly been on treating petitioner's stroke." (ECF No. 69, p. 7, n. 6.)

[11] Petitioner neglected to file a CV for Dr. Gershwin in this case. However, Dr. Gershwin does include some summary of his relevant qualifications in this initial report. (Ex. 10, p. 1.) In any event, Dr. Gershwin is well-known to the program and respondent has not argued that Dr. Gershwin is unqualified to offer the opinion he has provided in this case. (ECF No. 69.) For an example of a prior decision citing the qualifications contained within Dr. Gershwin's CV, *see Sturdevant v. Sec'y of Health & Human Servs.*, No. 17-172V, 2022 WL 3369716, at *6 (Fed. Cl. July 19, 2022) (special master Dorsey discussing Dr. Gershwin's qualifications in a Bell's Palsy case).

with Bell's Palsy. (*Id.* at 3.) The mechanisms involved in Bell's Palsy also remain "enigmatic," but Dr. Gershwin explains that the damage arises from inflammation of the facial nerve with subsequent compression and permanent damage to the nerve. (*Id.* at 2.) While the molecular mechanisms of Bell's Palsy have not been dissected, according to Dr. Gershwin, the mechanisms involved in this disorder appear to be a "highly focal inflammatory response." (*Id.* at 3.) Dr. Gershwin suggests immunization is one possible putative etiology of Bell's Palsy; and notes reports of Bell's Palsy post influenza and hepatitis B vaccination. (*Id.* (citing Barbara Rath et al., *"All that palsies is not Bell's" – The need to define Bell's palsy as an adverse event following immunization,* 26 VACCINE 1 (2007) (Ex. 10.11); Ali Rowhani-Rahbar et al., *Immunization and Bell's Palsy in Children: A case-centered analysis,* 175(9) AM J. EPIDEMIOL. 878 (2012)(Ex. 10.12)).)

Dr. Gershwin opines that following the flu vaccine, petitioner's innate immune response included a "localized reaction within the trigeminal nerve, similar to the mechanisms associated with a viral infection induction of Bell's Palsy." (Ex. 10, p. 3.) Following an influenza vaccine, individuals produce both an innate and an adaptive response. (*Id.*) In petitioner's case, there wouldn't be evidence of an adaptive response. (*Id.*) Instead, Dr. Gershwin points to a "tissue-specific" innate response that is "unique to individuals genetically susceptible to trigeminal neuralgia." (*Id.*) Additionally, Dr. Gershwin remarks that "in every immune response, there is genetic variation, including examples of exaggerated innate or adaptive response." (*Id.*) Dr. Gershwin emphasizes this variation because it may "lead to rare events that would be below the level of detection of epidemiological analysis." (*Id.*)

According to Dr. Gershwin, petitioner received an influenza vaccine which caused a focal innate response, which produced edema, and ultimately compromised petitioner's trigeminal nerve. (Ex. 10, p. 4.) Lastly Dr. Gershwin cites one report of trigeminal neuralgia following an influenza vaccination in 1985. (*Id.* (citing Michael Demmler & Gerd Heidel, *Trigeminal disorder after influenza vaccination,* 37 PSYCHIAT. NEUROL. MED. PSYCHOL. 428 (1985) (Ex. 12.2).) He notes that petitioner's prior history of migraine headaches, diabetes, and her abnormal MRI with plaques interpreted as MS do not undermine his opinion, "as these features are not etiological [*sic*] associated with trigeminal neuralgia." (*Id.*)

In his first supplemental report, Dr. Gershwin acknowledges that Bell's Palsy and trigeminal neuralgia are "entirely different syndromes." (Ex. 11, p. 1.) Dr. Gershwin suggests that his comparison of the two diseases demonstrates the similar "damage" that occurs from inflammation and that in both cases "inflammation leads to compression." (*Id.*) Dr. Gershwin maintains that Bell's Palsy is an autoimmune response and "[t]he pathology is thought to be an inflammatory response." (*Id.* (citing A. Greco et al., *Bell's palsy and autoimmunity,* 12 AUTOIMMUNITY REV. 323 (2012) (Ex. 11.1)).) Dr. Gershwin explains that few, if any, pathological studies exist that examine the role of cytokines and the inflammatory response on trigeminal neuralgia. (Ex. 11, p. 2.) Dr. Gershwin criticizes Dr. Sriram's report because it "ignores the genetic individual differences between hosts and does not offer an alternative mechanistic explanation."

(*Id.*) Because trigeminal neuralgia is rare, Dr. Gershwin contends that it will not be detected by the traditional epidemiological analyses conducted for the flu vaccine. (*Id.*)

Dr. Gershwin opines that petitioner suffered a "simple anatomic inflammatory obstruction," rather than a systemic reaction. (*Id.*) He adds that it is not unusual for an individual to have lymphadenopathy in their upper extremities following a deltoid immunization. (*Id.*) Dr. Gershwin cites a study concerning how the macrophage death following influenza vaccination initiates an inflammatory response that promotes dendritic cell function in the draining lymph node, which he suggests illustrates the "local response" which is "the site in which innate immunity is initiated that will lead to localized swelling." (*Id.* (citing Nikolaos Chatziandreou et al., *Macrophage death following influenza vaccination initiates the inflammatory response that promotes dendritic cell function in the draining lymph node,* 18 CELL REP. 2427 (2017) (Ex. 11.2)).)

In his second supplemental report, Dr. Gershwin explains that the reaction to the vaccine is dependent upon the lymphatic system, the local anatomy, and where lymph nodes drain. (Ex. 12, p. 2.) The lymphatic system removes fluid, including plasma proteins, cells and cellular debris, which ultimately end up in the venous system. (*Id.*) According to Dr. Gershwin, this explains why an individual may receive a vaccination in the right deltoid, for example, but suffer swelling in their cervical lymph nodes, on both sides of the body. (*Id.*) While "there is no etiological event that [Dr. Gershwin] can identify within the medical records that would have led to trigeminal neuralgia," he opines petitioner's immunization "should have produced an immune reaction." (*Id.*) Dr. Gershwin concludes petitioner's trigeminal neuralgia is "clearly a rare event," but "rare events do occur and should not be discounted." (*Id.*)

b. Respondent's Expert, Subramaniam Sriram, M.D.

Dr. Sriram received his M.B., B.S. from the University of Madras, India in 1973. (Ex. B.) He completed a Neurology residency and post-doctoral fellowship at Stanford University. (*Id.*) Dr. Sriram currently holds a teaching position as a professor in neurology and microbiology immunology. (Ex. A.) Additionally, Dr. Sriram is the director of the Multiple Sclerosis (MS) Clinic at Vanderbilt Medical Center, where he performs research on the causes and treatment of MS and cares for over 1,000 MS patients. (*Id.* at 1.) Among his patients Dr. Sriram treats a subset that also suffer from trigeminal neuralgia. (*Id.*) He is board-certified in internal medicine and neurology and authored many publications on MS. (*Id.*)

Dr. Sriram opines that petitioner likely suffered from both trigeminal neuralgia and Hemi facial spasms. (Ex. A, p. 2.) Dr. Sriram emphasizes that the etiology in most cases of trigeminal neuralgia, "if not the majority," is compression of a tortuous artery in the posterior fossa, impinging on the fifth cranial nerve. (*Id.*) This vascular abnormality, Dr. Sriram explains, is more often seen in patients with cerebrovascular disease and hypertension, both of which were observed in petitioner. (*Id.*) In approximately 15% of patients, a structural abnormality is observable in the posterior fossa. (*Id.* at 3.) While in a younger subset of patients with MS, Dr. Sriram notes that trigeminal neuralgia is

due to "a demyelinating lesion in the root entry zone of the sensory division of the Trigeminal nerve." (*Id.*)

Dr. Sriram notes that the pathophysiology of Hemi facial spasms is similar to that of trigeminal neuralgia. (Ex. A, p. 3.) The etiology of Hemi facial spasms, however, is a vascular anomaly of the vertebral artery which impinges on the seventh cranial nerve. (*Id*.) This disorder results in frequent and episodic contraction of the facial muscles. (*Id.*) While both disorders are well known, Dr. Sriram suggests that ipsilateral coexistence of Hemi facial spasms and trigeminal neuralgia among patients is not uncommon. (*Id.*) The coexistence of these two disorders is commonly referred to as "*tic convulsif*;" and Dr. Sriram notes that since 1920 approximately four dozen cases have been reported. (*Id.* (citing E.T. Fonoff et al., *Neurovascular compression in painful tic convulsif,* 151 ACTA NEUROCHIR. 989 (2009) (Ex. H)).) Some of these cases were caused by tumors in the posterior fossa, but the majority involved an ectatic vertebral artery. (*Id.*) Another study by Cook and Janetta reported 11 cases of *tic convulsif* treated by microvascular decompression of both cranial nerves V and VII. (*Id.* (citing Bruce R. Cook & Peter J. Jannetta, *Tic convulsif: results in 11 cases treated with microvascular decompression of the fifth and seventh cranial nerves,* 61 J. NEUROSURG. 949 (1984) (Ex. I)).)

Dr. Sriram opines that "there are no comparable similarities" between trigeminal neuralgia and Ball's Palsy. (Ex. A, p. 3.) The two diseases are "entirely different clinical syndromes." (*Id.*) Additionally, Dr. Sriram stresses that neither trigeminal neuralgia nor Bell's Palsy are immunological diseases. (*Id.*) Dr. Sriram further stresses that there are no studies or reports that demonstrate that: (1) the influenza vaccine causes a focal innate response in the cranial nerves; (2) an innate immune response to the cranial nerves causes edema and pain; or (3) that an innate immune response is the cause of either trigeminal neuralgia or Hemi facial spasms. (*Id.* at 3-4.) As a result, Dr. Sriram opines that petitioner's receipt of her influenza vaccine is not casually connected to trigeminal neuralgia or Hemi facial spasms. (*Id.* at 4.)

In his first supplemental report, Dr. Sriram again stresses petitioner suffers from both Hemi facial spasms and trigeminal neuralgia, more specifically *tic convulsif*. (Ex. DD, p. 2.) This distinction, according to Dr. Sriram, is not trivial. (*Id.*) Instead, it "points to the fact that the clinical symptoms arise most likely from ectatic dilatation either the vertebral artery or its branches." (*Id.*) Still, neither syndrome is inflammatory. (*Id.*) Dr. Sriram agrees that both Bell's Palsy and Hemi facial spasms are both neuropathy disorders. (*Id.* at 3.) However, he stresses that the term "neuropathy" does not indicate an underlying etiology (whether it be structural, traumatic, genetic, inflammatory, infectious, etc.). (*Id.*)

Additionally, Dr. Sriram notes that there are no lymph nodes in the posterior fossa at or in the course of the ophthalmic division of cranial nerve which would cause a compressive neuropathy or at the origin of cranial nerve VII, unlike Dr. Gershwin suggests. (Ex. DD, p. 3.) Dr. Sriram agrees that a subset of trigeminal neuralgia is inflammatory. (*Id.* at 4.) However, those cases involve patients with multiple sclerosis,

where inflammation at the root entry zone of cranial nerve 5 can cause trigeminal pain. (*Id.*) He stresses the majority of cases of trigeminal neuralgia and Hemi facial spasms, specifically among the elderly, result from ectatic dilation of cerebral arteries from atherosclerotic vascular disease. (*Id.*) Petitioner has diabetes, hypertension, hyperlipidemia, and disturbance of heart rhythm – all of which Dr. Sriram notes are indicative of diffuse atherosclerotic vascular disease and likely to include cerebral blood vessels. (*Id.*)

In his second supplemental report, Dr. Sriram asserts "Dr. Gershwin ignores the cause of [petitioner's] facial spasms entirely." (Ex. GG, p. 2.) Dr. Sriram contends that the innate immune response in the lymph nodes which drain the neck and throat is meaningless to the process that causes trigeminal neuralgia. (*Id.*) Anatomically speaking, Dr. Sriram stresses that the fifth cranial nerve is located in the brain, away from any draining lymph nodes. (*Id.*) Even the evidence of lymph flow in the brain that Dr. Gershwin presents does not "in any way offer [a] mechanism of activation of innate immune pathways sufficient for the development of TN and Hemifacial spasms." (*Id.*) Ultimately, Dr. Sriram asserts that without an angiogram of petitioner's blood vessels in the posterior fossa, one cannot opine on the etiological event that led to petitioner's trigeminal neuralgia. (*Id.*)

Lastly, Dr. Sriram suggests that the single incidence of trigeminal neuralgia post flu vaccination that Dr. Gershwin relies upon is distinguishable. (Ex. GG, p. 3.) That patient complained of "a weird feeling of the face;" pressure pain in front of the right ear and in the lateral nasal area; blurred vision of the right eye and retro-bulbar pressure; and general fatigue and weakness. (*Id.*) The nature of that flu vaccination was not reported, and Dr. Sriram emphasizes that "at no point in the case report do the authors state that [the] patient had [trigeminal neuralgia], nor do they refer to the development of Hemi facial spasms." (*Id.*)

c. Respondent's Expert, Neil D. Romberg, M.D.

Dr. Romberg received his medical degree from Pennsylvania State College of Medicine in 2004. (Ex. D.) He completed his residency at New York University and was an immunology fellow at Yale University. (*Id.*) Dr. Romberg is currently an assistant professor of Pediatrics at the University of Pennsylvania and an attending physician at the Children's Hospital of Philadelphia. (Ex. C.) Dr. Romberg also serves as the Jeffrey Modell Endowed Chair for Pediatric Immunology Research. (*Id.*) Dr. Romberg's research focuses on the failure of immunologic tolerance, autoantibody production, and excessive activation of the innate immune system. (*Id.*) He is board certified in pediatrics and allergy / clinical immunology. (*Id.*) Dr. Romberg notes that he is not a neurologist and limits his opinions to the immunological aspects of clinical neurology. (*Id.*)

Dr. Romberg likewise agrees that there is no evidence that the adaptive immune system contributed to petitioner's development of trigeminal neuralgia. (Ex. C, p. 3.) Dr. Romberg first explains that most vaccines are designed to activate the innate

immune system using either adjuvant alum or MF59, although prior to 2015 the seasonal influenza vaccine was non-adjuvated (and less likely to cause local inflammation). (*Id.* at 3.) The key question, according to Dr. Romberg, is whether petitioner's innate immune response was unintentionally excessive. (*Id.*) If that is the case, then whether there is evidence of systemic inflammation that damaged petitioner's trigeminal nerve. (*Id.*) Dr. Romberg stresses that petitioner's medical records present no evidence that petitioner experienced a visible local reaction at the vaccination site. (*Id.* at 4.) Nor was there evidence that petitioner experienced any systemic inflammatory symptoms – based on her concentrations of IL-1beta, TNF-alpha, GMCSF and IL-6. (*Id.*)

Dr. Romberg acknowledges that a well-contained inflammation within local tissues may not induce systemic symptoms, but if that were the case for petitioner's left trigeminal nerve, it is unlikely that a seasonal influenza vaccine could induce inflammation at a location so remote from the injection site. (Ex. C, p. 4.) Local spread of inflammation through soft tissues, observable through visible induration, was not reported. (*Id.*) Dr. Romberg asserts that spreading through small blood vessels is "nonsensical" because the blood supply to-and-from the left deltoid is not shared with the trigeminal nerve and gravity drains the deltoid lymphatics "down to axillary lymph nodes and not up to the skull." (*Id.*)

Dr. Romberg contends trigeminal neuralgia is not an inflammatory disease, except in cases of coexisting multiple sclerosis. (Ex. C, p. 4.) Dr. Romberg agrees with Dr. Sriram, finding that most cases of trigeminal neuralgia are caused by physical compression of the cranial nerve. (*Id.*) And for this reason, Dr. Romberg finds that "it is not surprising that vascular diseases like hypertension, diabetes, and headaches, all diagnoses made in [petitioner], are risk factors for developing trigeminal neuralgia. (*Id.*) Dr. Romberg likewise agrees that trigeminal neuralgia is not analogous to Bell's Palsy. (*Id.*) While there are many indications that Bell's Palsy is a post-infectious inflammatory disease, the same cannot be said for trigeminal neuralgia. (*Id.*)

Dr. Romberg likewise recognizes the possibility of a genetic predisposition to trigeminal neuralgia. (Ex. C, p. 5.) However, Dr. Romberg notes that there are no documented cases of trigeminal neuralgia in petitioner's family and familial cases of this disorder are associated with "vascular cases that do not involve the immune system." (*Id.*) Lastly, Dr. Romberg opines that even if trigeminal neuralgia was the presenting feature of underlying multiple sclerosis, the injury would extend directly from petitioner's central nervous system and not from her left deltoid vaccine injection site. (*Id.*)

In his first supplemental report, Dr. Romberg agrees that lymphedema, or obstruction of lymphatic vessels, may occur post-vaccination. (Ex. EE, p. 2.) This causes swelling in the affected extremity. (*Id.*) However, Dr. Romberg notes that no swelling was recorded in petitioner's case. (*Id.*) Nonetheless, obstruction of the lymphatic vessels would contradict Dr. Gershwin's theory. (*Id.*) He explains that lymphedematous obstruction limits, rather than encourages, the flow of lymph out of the immunized extremity. (*Id.*) This, according to Dr. Romberg, undermines Dr. Gershwin's

theory that the influenza vaccine spread from the deltoid to the trigeminal nerve. (*Id.*) Lastly, while ipsilateral lymphadenopathy may occur post-vaccination, contralateral lymphadenopathy is unexpected because the lymphatic drainage is centripetal, not centrifugal. (*Id.*)

In his second supplemental report, Dr. Romberg explains the lymphatic drainage of the deltoid tissues moves into the deltopectoral lymph nodes, as well as the infraclavicular and axillary lymph nodes, before entering venous circulation via the thoracic duct. (Ex. HH, p. 1.) None of these lymph nodes are proximate to the trigeminal nerve, and Dr. Romberg stresses that all of them drain in the opposite direction toward the vena cava. (*Id.*) Dr. Romberg notes that in the single report of trigeminal neuralgia post flu vaccination the authors were reluctant to draw a causal relationship, stating that "the criteria for a 'reliable' assignment were not met." (*Id.* at 2 (quoting Demmler & Heidel, *supra,* at Ex. 12.2).) In this case, Dr. Romberg points to petitioner's pre-existing hypertension, diabetes, and headaches as major risk factors for trigeminal neuralgia. (Ex. HH, p. 2.)

Dr. Romberg also authored a report in response to petitioner's supplemental medical literature, Exhibits 13-17. (Ex. JJ; *See* ECF No. 34.) Dr. Romberg notes that Exhibits 13-16 do not mention the trigeminal nerve nor trigeminal neuralgia. (Ex. JJ, pp. 2-3.) Dr. Romberg likewise agrees with the Pal and Ramsey study: lymph travels in a "one-way anterograde fashion from capillary beds centripetally into venous circulation." (*Id.* at 2; Ivy Pal & Joshua D. Ramsey, *The role of the lymphatic system in vaccine trafficking and immune response,* 63 ADV. DRUG DELIV. REV. 909 (2011) (Ex. 13).) However, Dr. Romberg stresses that this contradicts Dr. Gershwin's theory that elements of the innate immune system traveled centripetally from the left deltoid and centrifugally *upward* to the trigeminal nerve. (*Id.*) As Dr. Romberg explains, the Mallick and Bodenham study describes how interruption of lymphatic vessels via surgical ligation or parasitic infection may cause lymphedema. (*Id.*) Yet, Dr. Romberg notes that there is no indication in petitioner's medical records of a surgical procedure or parasitic infection that may have interrupted lymph flow. (*Id.*) Nor is there mention of a physical finding consistent with lymphedema. (*Id.*)

Dr. Romberg quotes the Moore and Bertram study for the conclusion that lymphatics drain "'every part of the body except the brain and spinal cord'" – locations crucial to petitioner's theory of causation. (Ex. JJ, p.3 (quoting James E. Moore Jr. & Christopher D. Bertram, *Annual Review of Fluid Mechanics: Lymphatic system flows,* 50 ANN. REV. FLUID MECH. 459 (2018) (Ex. 15)).) Even the "alternative clearance system" proposed by Moore and Bertram for the brain and spine follows the same conventional lymphatics—flowing centripetally. (Ex. JJ, p. 3.) According to Dr. Romberg, the McDonald study similarly defeats Dr. Gershwin's theory of retrograde lymphatic flow. (*Id.*) Author Susan Kratz discusses trigeminal neuralgia, which she theorizes is caused by a disorder of an alternative lymphatic system. (*Id.*) According to Dr. Romberg, this theoretical system drains the central nervous system; and while her findings "may challenge conventional views," Dr. Romberg stresses that this alternative system does not support petitioner's theory of causation. (*Id.*) Ultimately Dr. Romberg concludes

petitioner's supplemental literature "reinforce the conventional wisdom that lymphatic drainage is one-way anterograde and centripetal." (*Id.*)

V. Discussion

a. *Althen* prong one

Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.,* 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (citations omitted). To satisfy this prong, petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549. However, petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325–26 (Fed. Cir. 2006)). Scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Nonetheless, although petitioners cannot be *required* to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect" (*Capizzano*, 440 F.3d at 1325), the special master may consider and evaluate such evidence when filed. *Andreu.,* 569 F.3d at 1379 (Special masters may consider medical literature and epidemiological evidence, when it is submitted, in "reaching an informed judgment as to whether a particular vaccine likely caused a particular injury.").

In this case Dr. Gershwin opines that lymph node macrophages process vaccine antigens creating a cytokine response, resulting in localized, anatomic inflammatory obstruction. (ECF No. 68, pp. 9-10; Exs. 11, 12.) He further opines that edema from draining lymph nodes surrounding the face, neck, scalp could have compressed the trigeminal nerve. (*Id.*) Dr. Gershwin seeks to derive support for his contention that trigeminal neuralgia can be linked to vaccination through three points, none of which are persuasive. First, he opines that trigeminal neuralgia can be analogized to Bell's Palsy due to the fact that both conditions are neuropathies affecting facial nerves. He acknowledges that in contrast to trigeminal neuralgia where direct evidence of either cytokine-response or vaccine-causation is virtually absent (Ex. 11, p. 2), there is evidence available to potentially link Bell's Palsy to vaccination as a consequence of an inflammatory process. Second, he indirectly references an article later filed by petitioner without comment regarding the efficacy of craniosacral therapy in the treatment of trigeminal neuralgia. The implication seems to be that the study supports a hypothesis that trigeminal neuralgia may relate to inflammation in lymphatic vessels surrounding the brain, which would potentially bring the posited edema into anatomic

proximity to the nerve at issue. And third, he presents an article presenting a case report of trigeminal neuralgia following influenza vaccination.

Broadly speaking, Bell's Palsy and trigeminal neuralgia are both neuropathy disorders as Dr. Gershwin suggests, but, as Dr. Sriram explains, the term "neuropathy" does not identify an underlying etiology—whether that be structural, traumatic, genetic, inflammatory, or infectious. (Ex. DD, p. 3.) Dr. Gershwin opines both Bell's Palsy and trigeminal neuralgia are caused by inflammation that leads to compression. (Ex. 11, p. 1.) However, this is broad and imprecise. Bell's Palsy is an idiopathic peripheral nerve palsy involving the facial nerve (cranial nerve VII). (Greco et al., *supra,* at Ex. 11.1; Rath et al., *supra,* at Ex. 10.11.) The facial paresis, or paralysis, is thought to result from facial nerve inflammation, consistent with an infectious or immune cause. (Ex. 11, p. 1 (Dr. Gershwin's report) ("The pathology [of Bell's Palsy] is thought to be an inflammatory response"); Rath et al., *supra,* at Ex. 10.11, p. 6; Greco et al., *supra,* at Ex. 11.1, p. 3.) According to Dr. Gershwin, a leading theory of Bell's Palsy is autoimmune demyelination leading to demyelination of the facial nerve. (Ex. 11, pp. 1-2.) To the extent Bell's Palsy additionally involves compression, it results from the inflamed nerve becoming entrapped by the narrow bony Fallopian canal through which it runs. (Rath et al., *supra,* at Ex. 10-11, p. 6.) In contrast, trigeminal neuralgia is generally believed to relate to vascular compression. (*E.g*. Luke Bennetto et al., *Trigeminal neuralgia and its management,* 331 BMJ 1, 1-2 (2007) (Ex. 10.1); Eller et al., *supra,* at Ex. 10.2, p. 1.) It is also commonly seen as accompaniment to facial spasms in the context of tic convulsif, a broader syndrome also associated with nerve compression which can also be due to vascular or other structural abnormalities. (Fonoff et al., *supra,* at Ex. H, p. 2 (citing Cook & Jannetta, *supra,* at Ex. H); Love & Coakham, *supra,* at Ex. 10.4; Zakrzewska & McMillan, *supra,* at Ex. E, p. 1 ("[t]hose with symptomatic TN have either a compression of the trigeminal nerve caused by tumours (benign and malignant) or other structural abnormalities such as arteriovenous malformations, or have multiple sclerosis (MS)).) To the extent demyelination is sometimes seen in trigeminal neuralgia, it is considered sequala of the compression itself rather than evidencing an autoimmune process. (Eller et al., *supra,* at Ex. 10.2, p. 1.) In some cases, trigeminal neuralgia may also be caused by facial trauma, oral or facial surgery, tumors, or multiple sclerosis.[12] (Susan Vaughan Kratz, *Manual Therapies reduce pain associated with trigeminal neuralgia,* 1(1) J. PAIN MGMT THER. 1 (2016) (Ex. 17).) Some studies have suggested that a minority of Bell's Palsy cases may present with symptoms of trigeminal nerve deficits, but other studies have found those symptoms may exist without involvement of the trigeminal nerve. (Rath et al., *supra,* at Ex. 10-11, p. 5.)

Based on my review of the literature filed in this case, Dr. Gershwin is alone in directly analogizing Bell's Palsy to trigeminal neuralgia. In fact, Dr. Gershwin has cited

[12] Even in the cases of overlapping MS, the literature describes a "double crush mechanism," namely, "inflammatory demyelination and *mechanical* demyelination on the same first-order neurons." (Truini et al., *supra,* at Ex. U, p. 4 (emphasis added).) To be clear, Dr. Gershwin does not opine that petitioner had coexisting MS. (*See* Exs. 10, 11, 12.)

to one study by Rath, et al., the very purpose of which seems to be to caution against Dr. Gershwin's own approach to this case. That study, titled "'All that palsies is not Bell's' – The need to define Bell's palsy as an adverse event following immunization," explains that the precise definition of Bell's Palsy remains controversial and that this has hindered exploration of the underlying causal mechanisms. (Rath et al., *supra,* at Ex. 10.11.) The authors specifically stress that creating a workable definition for Bell's Palsy is a requirement for assessing Bell's Palsy as a post-vaccination phenomenon. (*Id*. at 7.) Among many other points discussed, the article explains that Bell's Palsy is a diagnosis of exclusion and that trigeminal neuropathy is among a long list of other conditions that must be excluded to reach a valid diagnosis. (*Id*. at 6 (Table 4).) Indeed, it is not even clear from Dr. Gershwin's own reports why he would even invoke the analogy. When pressed on the question, Dr. Gershwin stresses that Bell's Palsy is an inflammatory condition, but also discusses it as a potential autoimmune condition leading to demyelination akin to being a subtype of GBS. (Ex. 11, pp. 1-2.) However, this bears no obvious relation to his theory of an innate immune response that would lead to nerve compression due to lymph node swelling.

The previously presiding special master gave petitioner an opportunity to cure this shortcoming. Specifically, after reviewing Dr. Gershwin's first two reports, the special master "expressed concern that, other than both conditions being neuropathic and affecting the face, it is not clear that Bell's Palsy and trigeminal neuralgia are related based on Dr. Gershwin's reports." (ECF No. 28.) Petitioner's counsel agreed that further information on that point would be helpful. (*Id*.) However, Dr. Gershwin's subsequently filed report focused exclusively on the inflammatory nature of Bell's Palsy without a single word substantiating his assertion that trigeminal neuralgia similarly results from an inflammatory process. (Ex. 12.) The special master then held a further status conference and advised that Dr. Gershwin had not answered her question. (ECF No. 33.) Petitioner indicated she would file additional literature to further explain the theory of causation. (*Id*.) However, I have reviewed the literature subsequently filed by petitioner (ECF No. 34; Exs. 13-17) and only one of the articles addresses trigeminal neuralgia at all.

That article is from the Journal of Pain Management and Therapy, authored by Susan Vaughan Kratz. Petitioner appears to present it as evidence that trigeminal neuralgia is caused by a disorder of an alternative lymphatic system. This literature was filed without commentary or analysis from Dr. Gershwin. (*See* ECF Nos. 33-34.) Kratz's article summarizes the outcomes of three adults receiving manual therapies to treat trigeminal neuralgia. (Kratz, *supra,* at Ex. 17.) The author is an occupational therapist with experience in neurological and general rehabilitation. (*Id.* at 3.) The paper discusses "emerging theories" that could suggest a relationship between idiopathic nerve pain affecting the trigeminal nerve and an "alternative" lymphatic system that may facilitate drainage from the central nervous system (otherwise thought

not to have lymphatic draining) via lymphatic vessels in the dura mater.[13] Given this hypothesis, the purpose of the paper was to explore whether craniosacral therapy may be a viable treatment option if it can improve fluid exchange and enhance drainage of this alternative lymphatic system. (*Id*. at 5.) Importantly, however, the author acknowledges that the efficacy of such treatment is unclear and this study, which had only three subjects, had mixed results. (*Id*.)

If petitioner's theory were specifically based on this alternative lymph system (which would not be obvious from Dr. Gershwin's reports), the Kratz paper would still only provide very little evidence. However, that is not what Dr. Gershwin discusses in his reports. Whereas this paper appears to theorize that central nervous system inflammation may affect lymphatic tissue in the dura mater hypothesized as an "alternative" lymphatic system, Dr. Gershwin posits that inflammation originating from the extremity might result in inflammation via the traditionally understood lymphatic system within a collection of lymph nodes around the face, neck, and scalp known as "Waldeyer's ring." (Ex. 12, p. 2.) He only cryptically seeks to extend this suggestion further by noting merely the fact of papers identifying lymphatic vessels in addition to lymph nodes without further explanation. (*Id*.) Additionally, respondent's expert presentation stresses conventional wisdom that lymphatic drainage is one-way anterograde and centripetal. (*See* Ex. JJ, pp. 3-4; Pal & Ramsey, *supra,* at Ex. 13; Moore & Bertram, *supra,* at Ex. 15; A.J. MacDonald et al., *Modeling flow in collecting lymphatic vessels: one-dimensional flow through a series of contractile elements,* 295 AM. J. PHYSIOL. HEART CIRC. PHYSIOL. 1 (2008) (Ex. 16); Aspelund et al., *supra,* at Ex. KK.) Dr. Gershwin does not explain how his theory addresses that conventional wisdom and nothing in the Kratz article, or the literature filed by petitioner, otherwise appears to refute it. Nor does the Kratz paper otherwise provide any evidence to suggest that trigeminal neuralgia can be understood to have an inflammatory etiology

[13] In relevant part, Kratz notes that the brain lacks a lymphatic circulation system and thus must clear extracellular protein by some alternative mechanism. (Krtaz, *supra,* at Ex. 17, p. 2.) Kratz concedes "[t]hough it is unknown how solutes from the brain interstitium move between the parenchyma to the cerebral spinal fluid (CSF)," one study she cites shows that CSF enters the parenchyma along "perivascular spaces" that surround penetrating arteries, while brain interstitial fluid is cleared along perivenous drainage pathways—essentially supposing the perivascular space serves as a lymphatic system equivalent for the brain. (*Id.*) From this study, Kratz proposes the "lack of movement of noxious fluids in the synaptic spaces may contribute to the inflammatory environment leading to idiopathic nerve pain." (*Id.*) Kratz cites an article by Aspelund et al. (subsequently filed by Dr. Romberg), also describing the presence of lymphatic vessels in the dura mater *of mice,* where fluid injected into these vessels drained out of the CNS and into deep cervical lymph nodes at the base of the skull. (Aleksanteri Aspelund et al., *A dural lymphatic vascular system that drains brain interstitial fluid and macromolecules,* 212(7) J. EXP. MED. 991 (2015) (Ex. KK).) However, Dr. Romberg explains that "[a]lthough these nodes physically communicate with the conventional lymphatic system, the described murine lymph vessels at the skull's base posses the same one-way vales that conventional lymphangions use to prevent retrograde lymph flow." (Ex. JJ, p. 3.) Again, as in the traditional lymphatic systems, this system described by Aspelund et al., describes anterograde, centripetal lymphatic flow.

comparable to Bell's Palsy. Thus, the Kratz paper does not appear to support petitioner's case.

To be sure, there are several disorders associated with abnormal lymph flow. (Moore & Bertram, *supra,* at Ex. 15.) In that regard Dr. Gershwin opines that it is not unusual for an individual to have lymphadenopathy in their upper extremities following a deltoid immunization. (Ex. 11, p. 2.) Dr. Romberg agrees that lymphedema, or obstruction of lymphatic vessels, may occur post-vaccination to cause swelling in the affected extremity, which is not the scenario in this case. (Ex. EE, p. 2.) However, he opines that obstruction of the lymphatic vessels would actually contradict Dr. Gershwin's theory. (*Id.*) Lymphedematous obstruction limits, rather than encourages, the flow of lymph out of the immunized extremity. (A. Mallick & A. R. Bodenham, *Disorders of the lymph circulation: their relevance to anaesthesia and intensive care,* 91(2) BRIT. J. ANAESTHESIA 265 (2003) (Ex. 14).) This would undermine Dr. Gershwin's theory that the influenza vaccine spread from the deltoid to the trigeminal nerve via the lymphatic system. Mallick and Bodenham's study, offered by petitioner, describes how interruption of lymphatic vessels via surgical ligation or parasitic infection may cause lymphedema. (*Id.*) Yet, there is no indication in petitioner's medical records of a surgical procedure or parasitic infection that may have interrupted lymph flow. Nor is there mention of a physical finding consistent with lymphedema.

Without resort to evidence pertaining to Bell's Palsy to relate trigeminal neuralgia to vaccination, Dr. Gershwin is left with only a single case report from 1985 of a patient who suffered a "weird feeling on the face" and headaches two days post flu vaccination in 1982. (Demmler & Heidel, *supra,* at Ex. 12.2.) Sinusitis was excluded by the ENT side. (*Id.* at 3.) The patient additionally reported spontaneous and pressure pain in front of the right ear and in the lateral nasal area; occasionally this area was reddened. (*Id.*) Also, the patient complained of blurred vision of the right eye as well as retro-bulbar pressure and general fatigue and weakness. (*Id.*) Demmler and Heidel note the patient suffered prior "nerve inflammation" and was hospitalized for 4 weeks after suffering a viral flu infection in 1954. (*Id.*) The patient also suffered a "12-week local-type reaction" post influenza vaccination in 1981. (*Id.*) No further details of this reaction were provided. At no point in the report do the authors indicate this patient was diagnosed with trigeminal neuralgia specifically. Indeed, the authors explain that most cases of isolated cranial nerve disorders occur as part of a broader syndrome, but do not identify any specific relevant syndrome for assessment of their case. (Demmer & Heidel, *supra,* at Ex. 12.2, p. 4.) Still, the authors suggest there was a temporal relationship between the patient's vaccination and disease. (*Id.*) As with many atypical vaccination courses, "the exact evidence of the damage cannot be provided." (*Id.*) Nonetheless, Demmer and Heidel conclude "this atypical vaccination course [is] causally 'probable' since the incubation period and clinical picture corresponded to the diseases described in the literature" while noting the criteria for a "reliable" assignment were not met. (*Id.*)

Case reports, generally, "'do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value' . . . [but] 'the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary weight.'" *Paluck v. Sec'y of Health & Human Servs.*, 104 Fed. Cl. 457, 475 (2012) (quoting *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011), *aff'd*, 786 F.3d 1373 (Fed. Cir. 2015)). Case reports often present a detailed report of symptoms, signs, diagnosis, treatment, and follow-up care. Oftentimes petitioners in the Program will highlight the usefulness of case reports in cases of novel, unusual or rare diseases. *See Patton v. Sec'y of Health & Human Servs.*, 157 Fed. Cl. 159, 166-67 (2021). *But see Crutchfield v. Sec'y of Health & Human Servs.*, No. 09-39V, 2014 WL 1665227, at *19 (Fed. Cl. Spec. Mstr. Apr. 7, 2014) ("single case reports of Disease X occurring after Factor Y . . . do not offer strong evidence that the *temporal* relationship is a *causal* one—the temporal relationship could be pure random chance"), *aff'd*, 125 Fed. Cl. 251 (2014). Here, however, the Demmler and Heidel case report admittedly relies upon only the temporal association between the patient's vaccination and disease. (Demmer & Heidel, *supra,* at Ex. 12.2, p. 4.) The report broadly discusses neurological adverse reactions to the flu vaccine, including the notion of various isolated cranial nerve syndromes, but without identifying any diagnostically applicable syndrome for their subject. This reduces the light it could potentially shed on Dr. Gershwin's a theory of causation that the flu vaccine can cause trigeminal neuralgia. In fact, the case report authors do propose several possible mechanisms, none of which involve Dr. Gershwin's proposed lymphatic inflammation. (*Id*.) This case report is inadequate to carry petitioner's burden of proof, especially, but not only, because of the other shortcomings in Dr. Gershwin's broader explanation.

Taking all of this together, petitioner's claim that the flu vaccine can cause trigeminal neuralgia is not preponderantly supported. Dr. Gershwin offered an innate immune response with a localized reaction to the trigeminal nerve to explain how the vaccine resulted in petitioner's persistent facial spasming. However, the theory proposed by Dr. Gershwin relies upon a similarity between two different disorders, Bell's Palsy and trigeminal neuralgia, which is not supported. The fatal flaw with this proposed comparison is that trigeminal neuralgia is generally caused by neurovascular compression, not inflammation and/or autoimmune demyelination. Dr. Gershwin is also not persuasive in contending that the immune reaction beginning in petitioner's arm would lead to lymph node swelling that would cause nerve compression leading to trigeminal neuralgia. Thus, for all these reasons, petitioner has not met her burden under *Althen* prong one.

b. *Althen prong* two

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569

F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. *See* § 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed.Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed.Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs.*, 100 Fed.Cl. 119, 136 (2011), *aff'd*, 463 Fed. Appx. 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06–522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 Fed.Appx. 765 (Fed. Cir. 2012).

Petitioner relies heavily on the fact that "[t]here is nothing indicated in Petitioner's medical history that could have led to the development of trigeminal neuralgia." (ECF No. 68, p. 10.) She stresses that less than ten (10) percent of trigeminal neuralgia patients will have an identifiable cause other than a vascular compression lesion or multiple sclerosis. (Bennetto et al., *supra,* at Ex. 10.1, p. 2.) Accordingly, petitioner argues that, because she had neither vascular compromise nor multiple sclerosis, and because she received the flu vaccine at-issue in this case within three days of onset of her symptoms, she has demonstrated a logical sequence of cause and effect showing the flu vaccination was the cause of her trigeminal neuralgia. (ECF No. 68, pp. 10-11.) Certainly, petitioners in the Program are "permitted to use evidence eliminating other potential causes to help carry the burden on causation and may find it necessary to do so when the other evidence on causation is insufficient to make out a prima facie case." *Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1151 (Fed. Cir. 2007); *see also Pafford*, 451 F.3d at 1352. Here, however, the simple fact that petitioner's treating physicians did not identify a vascular compromise and petitioner did not suffer from multiple sclerosis is not compelling evidence that the vaccine more likely than not caused petitioner's condition. While petitioner's treating physicians did not find any

specific alternative explanation for her condition, they likewise did not conclude that her trigeminal neuralgia was vaccine-caused.

Instead, Dr. Sriram observes that petitioner's condition is better characterized as tic convulsif, which is the term coined for concurrent trigeminal neuralgia and hemifacial spasms. (Ex. A, p. 3 (citing Fonoff et al., *supra,* at Ex. H, p. 1; Cook & Jannetta, *supra,* at Ex. I, p. 1.).) Dr. Gershwin does not directly contest or even acknowledge Dr. Sriram's opinion that petitioner suffers from tic convulsif. (Ex. 11, p. 1 ("we all opine that the patient suffered from trigeminal neuralgia"); Ex. 12, pp. 1-3 (discussing trigeminal neuralgia).) However, Dr. Sriram stresses the distinction between trigeminal neuralgia and tic convulsif is not trivial. (Ex. DD, p. 2.) The presence of both trigeminal neuralgia and hemifacial spasms, according to Dr. Sriram, "points to the fact that the clinical symptoms arise most likely from ectatic dilatation of either the vertebral artery or its branches," and thus are not caused by inflammation. (Ex. DD, p. 2.)

Dr. Sriram bases his opinion that petitioner suffered tic convulsif on her complaints of facial spasms and facial pain. (Ex. A, p. 2.) He observes petitioner's pain was located in the forehead and rated as 20/10 pain. (*Id.* (citing Ex. 4, p. 5).) Although there is a description of pain along with the spasms, he concludes petitioner likely had "episodic contractions of muscles of the left side of the face along with pain around the orbit." (Ex. A, p. 2.) Dr. Sriram thus opines petitioner had both trigeminal neuralgia and hemifacial spasms. (*Id.*) In that context, imaging (MRI, CT scan) is necessary to rule out brain tumors, vascular malformations, aneurysms, and skull deformities. (*Id.*) Petitioner's MRI and CT scans did not reveal any brain tumors, vascular malformations, aneurysms, etc. (Ex. Ex. 4, p. 7; Ex. 6, pp. 74, 75; Ex. 8, p. 8; Ex. 20, p. 5.) However, Dr. Sriram contends that without an angiogram of petitioner's blood vessels in the posterior fossa one cannot definitively identify the pathomechanism for petitioner's tic convulsif.[14] (Ex. GG, p. 2.)

The development of tic convulsif in petitioner's case is consistent with the development of the condition generally. Dr. Sriram explains the tortuous vessels seen in cases of trigeminal neuralgia are commonly seen in patients with attendant risk factors—such as age, hypertension, and hyperlipidemia, all of which petitioner had. (Ex. GG, p. 2.) Over time, focal indentation and/or distortion of demyelinated axons leads to aberrant discharge of the nerve spontaneously, or in response to normally innocuous afferent traffic. (*Id.*; Eller et al., *supra,* at Ex. 10.2; Love & Coakham, *supra,* at Ex. 10.4.) Although, pain-free intervals are common and may continue "for weeks or as long as a few years." (Eller et al., *supra,* at Ex. 10.2, p. 1.) Thus, other than the temporal association to her vaccination, nothing in petitioner's medical records suggests the course of her tic convulsif was vaccine-caused. Often patients will continue to suffer

[14] However, I do not overlook the MRI and CT scans of petitioner's brain taken over the course of her treatment. In two of those scans, the findings indicate "unremarkable flow voids are demonstrated bilaterally at the base of the skull." (Ex. 4, p. 7 (MRI-brain with and without contrast on 1/12/2015); Ex. 20, p. 5 (MRI-brain without contrast on 11/9/2018).) Also, in petitioner's CT scan of the brain on November 3, 2016, the findings indicate "[n]o definite posterior fossa abnormality." (Ex. 8, p. 8.) Though, there is little to no record of Dr. Gadipudi, petitioner's treating neurologist, interpreting of these scans. (*See generally,* Exs. 18-21.)

intermittent pain and spasms until a nerve decompression is performed. (Love & Coakham, *supra,* at Ex. 10.4, pp. 7-8.) This can explain the continued trigeminal pain reported by petitioner over the course of approximately 5 years. (*See* Exs. 4, 18-22.)

Additionally, petitioner in this case apparently suffered a history of vascular compromise, in light of her history of atrial fibrillation, TIA, and stroke. (Ex. 3, pp. 1-4; Ex. 6, pp. 15-18, 23.) Diabetes is also a risk factor to the development of trigeminal neuralgia, as is a history of migraines. (*See, e.g.,* Pan et al., *Increased risk of trigeminal neuralgia after hypertension,: A population-based study,* 77 NEUROL. 1, 3 (2011) (Ex. Y) ("occurrence of hypertension was associated with a higher risk of developing TN"); Zhenq Xu et al., *Diabetes mellitus in classical trigeminal neuralgia: A predisposing factor for its development,* 151 CLIN. NEUROL. & NEUROSURG. 1, 2 (2016) (Ex. Z) ("Diabetes is a risk factor to the development of classical trigeminal neuralgia, and nerve damage [due] to hyperglycemia might be the linkage to the two diseases."); Kuan-Hsiang Lin et al., *Increased risk of trigeminal neuralgia in patients with migraine: a nationwide population-based study,* 36(13) CEPHALALGIA 1, 8 (2016) (Ex. AA) ("Migraine increases the relative risk of trigeminal neuralgia six-fold.").) These associations of other possible pathomechanisms for petitioner's tic convulsif cast further doubt on petitioner's contention that her condition was necessarily vaccine-caused.[15]

Petitioner's medical records also suggest that petitioner's treating neurologist more likely would agree with Dr. Sriram's assessment. While petitioner's medical records initially reflect a focus on trigeminal neuralgia (also referred to as tic douloureux) (Ex. 9, pp. 3-4; Ex. 4, p. 1-5), Dr. Gadipudi's records clearly indicate the presence of facial pain (i.e., trigeminal neuralgia) plus facial spasms (i.e., hemifacial spasms). (Ex. 4, pp. 1-6.) Moreover, on February 6, 2015, petitioner presented for follow-up with Dr. Gadipudi, with a chief complaint of left facial spasms. (Ex. 4, p. 4.) In the assessment, Dr. Gadipudi wrote, "[t]he patient was explained [*sic*] that the flu shot would not have any relation to the facial spasms." (*Id.*) Notably, in all of the follow-up visits with Dr. Gadipudi, no mention of her vaccination is made. (*See* Exs. 4, 18-22.) Dr. Gadipudi treated petitioner for her trigeminal pain and facial spasms from December 29, 2014, through November 1, 2019.[16] (*Id.*) Therefore, his denial of any logical sequence of cause and effect between petitioner's vaccination and her tic convulsif is compelling. Furthermore, I note that no other treating physician suspected petitioner suffered from an immune-mediated condition or response to vaccination that would otherwise support petitioner's theory of causation.

Overall, petitioner has not preponderantly proven that her flu vaccination did cause her trigeminal neuralgia, which is better understood as a component of tic convulsif. Though there is some suggestion of a temporal association, the opinions of petitioner's treating physicians and the overall course of her condition preponderantly

[15] Respondent, however, does not allege that any or all of these conditions represent an alternative cause that more likely than not caused petitioner's tic convulsif. (ECF No. 69, pp. 11-19.)

[16] No records from Dr. Gadipudi were filed for 2020 or 2021.

prove that her trigeminal neuralgia was consistent with the development of tic convulsif generally and without the need for the vaccination to act as an inciting event.

c. *Althen* prong three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed.Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed.Cl. 353 (2012), *aff'd mem.*, 503 Fed.Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

Here, petitioner has failed to meet her preponderant burden pursuant to *Althen* prongs one and two and thus she cannot prevail. In the interest of completeness, I note that the case report cited by Dr. Gershwin includes a list of prior instances of facial nerve disorder occurring post-influenza vaccination. That chart suggests an incubation period of between two to nine days (Demmler & Heidel, *supra,* at Ex. 12.2, p. 5.), which is consistent with the three-day onset in this case (Ex. 9, p. 4; Ex. 2, p. 16). Neither of respondent's experts have asserted any specific issue with regard to the timing of onset in this case. (*See* Exs. A, C, DD, EE, GG, HH, JJ.) Accordingly, had petitioner proven her case with respect to the first two *Althen* prongs, it possible that she may have prevailed with regard to *Althen* prong three.

VI. Conclusion

Petitioner has my sympathy for the injury she endured. Considering the record as a whole under the standards applicable in this Program, however, petitioner has not preponderantly established that her October 9, 2014, flu vaccination caused her condition. Accordingly, petitioner is not entitled to compensation. Therefore, this case is dismissed.[17]

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master

[17] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.